Visual Outcome in Herpes Zoster Optic Neuritis

A case report and systematic review

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Background: Optic neuritis in herpes zoster ophthalmicus (HZO) has been rarely reported in immunocompetent individuals. It is a potentially blinding disease. Visual outcomes for herpes zoster optic neuritis since the advent of acyclovir have only been mentioned through case reports. The role of systemic steroids is still unclear.

Objective: To present a case of herpes zoster ophthalmicus associated optic neuritis in an immunocompetent patient combined with systematic literature review to assess treatment outcomes.

Case: A 62-year-old male presents with left optic nerve edema concurrently with HZO. An MRI shows enhancement of the optic nerve consistent with optic neuritis. Pt is treated with systemic acyclovir and steroid with minimal visual acuity benefit. End result was light perception vision and optic nerve pallor.

Methods: An exhaustive MEDLINE literature search of cases of HZO optic neuritis in immunocompetent patients were reviewed and selected. Cases were excluded if the patient was immunocompromised, had extensive intracranial or orbital sequelae or in which sufficient information not available (n=2). Cases were separated by those having been treated with systemic acyclovir and those which were not. These were used to assess differences in visual prognosis based on mean visual acuity, recovery duration and treatment outcomes.

Results: A total of 26 cases met selection criteria, including this case. In the acyclovir treatment group there were 13 cases as well as 13 cases in the non-acyclovir treatment group. Both populations were comparable for gender and age at disease onset. The onset and recovery times were similar in each group. The mean visual acuity of the acyclovir treatment group was 0.66 logMAR compared to 1.38 in the non-acyclovir treatment group. This was statistically significant at P<0.005. Subset analysis of a systemic steroid only group and a systemic acyclovir + steroid group showed a mean logMAR visual acuity of 1.77 (n=6) and 0.7 (n=10) respectively. Legal blindness (logMAR 1.0) occurred in 23% of the acyclovir treatment group compared to 69% of the non-acyclovir treatment group. Optic atrophy was also more prevalent in cases not treated with acyclovir, 69%, compared to 31% if given systemic acyclovir.

Conclusion: Systemic acyclovir has dramatically reduced the morbidity of HZO optic neuritis. Although recovery time with the use of acyclovir was not significantly reduced, final visual acuity is much better when systemic acyclovir is implemented as primary treatment. Treatment with systemic acyclovir also reduced the rate of legal blindness and optic atrophy by almost 50%. The current treatment recommendations are systemic acyclovir for all cases of HZO. This will help reduce the ocular complications of HZO including HZO related optic neuritis.

Introduction

Herpes zoster is an infectious disease acquired from reactivation of the latent varicella virus. Reactivation can occur in individuals who had varicella virus infection “chicken pox” or received a vaccination for the virus earlier in life. The disease has an affinity for the nervous system in particular the sensory ganglia. The essential lesion is acute inflammation of these ganglion and nerves. Histological evidence show lymphocytic infiltration, hemorrhages, and fibrinoid necrosis of the infected ganglia, nerve cells and fibers. A lifelong inhabitance of the sensory ganglia occurs as a result of the initial infection and DNA evidence exists that confirms herpes zoster to be the reappearance of the same specific strain of childhood virus contracted years prior. It is estimated that over 90% of the population has se-
rologic evidence of the disease with over 1 million cases each year in the U.S. alone. It is a disease of the elderly with no seasonal pattern. As we age, we develop decreased cell-mediated immunity and it is by this mechanism that the virus is allowed to become active after having been held quiescent for many years.

This disease has a prodrome of fever, malaise, headache, and pain along the dermatome for a few days before the rash appears. The skin rash presents as an acute, painful, eruption of vesicles along a single dermatomic sensory distribution. If the virus reaches the globe via branches of the nasociliary nerve, it may cause ocular complications. New skin lesions typically cease formation by day 7, leaving stages of old crusting and fibrotic tissue.

HZO accounts for 17-50% of herpes zoster cases. Like herpes zoster, HZO is commonly reported in females although when HZO occurs early in life it occurs more commonly in males. The exanthem develops most commonly in the frontal branch of the ophthalmic nerve and does not cross the midline. It is potentially the most devastating form of herpes zoster due to its ocular complications which have a prevalence of up to 50%. It is well known that herpes zoster involving the nasociliary portion of the ophthalmic nerve carries an increased likelihood of ocular involvement. In one study, there was a 76% chance of ocular complications if the nasociliary nerve was involved compared to a 36% chance if it were not.

A 62 year-old male presented to our corneal clinic for evaluation of herpes zoster ophthalmicus. He is a previously healthy individual with no significant past medical history. Five days earlier he was diagnosed with conjunctivitis of his left eye which subsequently evolved into a vesicular rash distributed over the first trigeminal nerve. His eyelids had been...
come swollen shut and the eye had become increasingly painful.

On the fifth day he was able to open his left eye and noticed dramatically decreased vision. He was diagnosed with left herpes zoster ophthalmicus and referred to our clinic after starting acyclovir 800mg, five times per day. On exam, visual acuity was 20/25 in the right eye and 20/200 in the left. Pupils were equal with no relative afferent papillary defect (RAPD). Intraocular pressure (IOP) was significantly elevated on the left at 23 and 11 on the right. The vesicular rash covered the left V1 distribution of the trigeminal nerve, including the nasociliary branch. The left upper eye lid was erythematous and edematous and there were multiple margin lesions present. Slit lamp examination revealed 1+ injection of the conjunctiva and scattered diffuse pseudodendrites and edema of the cornea. Trace anterior chamber cellular reaction was present with normal iris and lens. Fundus ophthalmoscopy was normal with optic nerve cupping of 0.3 bilaterally. There were no significant laboratory abnormalities. General physical and neurological examinations were normal. The patient was started on topical steroid drops, four times per day as well as medication for intraocular pressure management, with follow up for the next day.

Six days after the onset of herpes zoster, the patient noticed a further decrease of visual acuity in the affected eye. He reported loss of central vision with preservation only at 11 o’clock. His visual acuity in the left eye had dropped to counting fingers (CF) and he now had a 2+ APD. The optic disc became hyperemic and edematous with blurred disc margins, and the patient was diagnosed with papillitis, most likely related to concurrent HZO.

This diagnosis was confirmed by neuro-ophthalmology who recommended visual field testing and MRI of the brain and orbits. Visual field testing of the left eye revealed a large central scotoma with minimal temporal sparing correlating with the patients’ symptoms (figure 1). The MRI results demonstrated enhancement of the left optic nerve, consistent with optic neuritis (figure 2). There were no other abnormalities noted on MRI testing. It was evident that our patient had optic neuritis secondary to herpes zoster ophthalmicus.

Four days after the initial consult, the patients vision had fallen to light perception (LP). He continued the directed course of acyclovir and began treatment with high dose systemic steroids. Two weeks afterward he reported mild improvement in his peripheral vision but his visual acuity remained at LP therefore the systemic steroids were discontinued.

One month after onset, examination demonstrated a 3+ APD and an IOP of 11 on the left. There were crusted lesions over the V1 distribution and mild left upper lid ptosis. The corneal pseudodendrites and anterior chamber inflammatory reaction had resolved. The optic nerve edema was replaced by diffuse nerve pallor. The patient was seen again at four months and his visual acuity remained at light perception with diffuse pallor of the optic nerve.

Figure 1. Visual field of the left eye showing dense central scotoma. Temporal sparing was noted. Right eye was normal.

Methods

An extensive search was performed of the MEDLINE database for cases of herpes zoster related optic neuritis in immunocompetent patients. Cases were reviewed and selected if adequate data could be extracted based on table 1. All types of optic neuritis were included namely, papillitis, neuroretinitis and retrobulbar optic neuritis. Cases were excluded that had extensive intracranial or orbital sequelae be-
cause this could potentially confound and contribute to the final visual outcome. Cases of immunocompromised patients and those with insufficient data were also excluded. Data extracted is shown in table 1 and included: age, sex, worst visual acuity, final visual acuity, onset of optic neuritis, estimated recovery from optic neuritis, findings, treatment, and end results.

Visual acuity measures from each report were converted to a standard logMAR equivalent for comparison. Visual acuities reported as count fingers (CF), hand motion (HM), light perception (LP) and no light perception (NLP) were assigned logMAR values of 2.0, 2.1, 2.2 and 2.3 respectively. These values were given in sequential order above the highest visual acuity reported in the selected cases which was 1.82. This allowed the possibility of certain statistical calculations across cases. Time was also converted to days in each case.

Cases were separated based on treatment and divided into 2 major groups; those treated with systemic acyclovir and those that were not. In each group, we calculated the mean visual acuity, time to onset, time to recovery and percentage of eyes that became legally blind (logMAR 1.0) or had optic atrophy. Subset treatment groups of systemic steroids only and systemic acyclovir + steroids were also formed from the data. These were assessed in the same manner with regards to visual acuity, onset and recovery.

**Results**

A total of 26 reported cases met inclusion criteria, including the one reported here. The baseline characteristics and findings are shown in table 1. There were 13 eyes included in the systemic acyclovir treatment group and 13 cases in the non-acyclovir treatment group. Both populations were comparable for gender and age at disease onset. The average age at disease onset was 53 and 58 years for the acyclovir and non-acyclovir treated groups respectively. These averages excluded the 5 reported cases that occurred in children. There were 7 females and 6 males in each group. The onset and recovery times were also similar in each group. Days to onset in the acyclovir group averaged 30 days with recovery occurring 151 days later. Onset was on average a few days earlier in the non-acyclovir treatment group at 23 days with recovery in 128 days (table 2).

Optic atrophy was assessed in each group occurring in a total of 14 of 26 eyes. In the acyclovir treatment group optic atrophy was the end result in 4 of 13 cases (31%) compared to 9 of 13 (69%) in the non-acyclovir treated group (table 2). Optic atrophy correlated well with visual acuity measures in each group.

The mean visual acuity of the acyclovir treatment group was 0.66 logMAR compared to 1.38 logMAR in the non-acyclovir treatment group (figure 3). This difference was statistically significant (P<0.005). Visual acuities in each group ranged from normal (0.0 logMAR) to NLP. Subset analysis of a systemic steroid only treatment group showed a mean

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**Figure 2.** A. Contrast-enhanced T1-weighted, fat-suppressed coronal MRI through the orbits shows contrast enhancement of the left optic nerve (arrow). B. Contrast-enhanced fat-suppressed axial MRI through the orbits shows contrast enhancement of the left optic (arrow).
Table 1. A comparison of literature reporting optic neuritis with herpes zoster ophthalmicus

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/ Sex</th>
<th>Worst V/A</th>
<th>Final V/A</th>
<th>Onset of ON</th>
<th>Estimated Recovery</th>
<th>Findings</th>
<th>Treatment</th>
<th>Final result</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veasey [31]</td>
<td>40/F</td>
<td>NLP</td>
<td>NLP</td>
<td>7 days</td>
<td>28 days</td>
<td>Central scotoma, Normal fundus</td>
<td>Mercurial inunctions, Pilocarpine sweats</td>
<td>Optic atrophy and central scotoma</td>
<td>1919</td>
</tr>
<tr>
<td>Jensen [32]</td>
<td>9/F</td>
<td>6/60</td>
<td>6/60</td>
<td>21 days</td>
<td>180 days</td>
<td>Disc edema, foveal exudates</td>
<td>Unknown</td>
<td>Optic atrophy</td>
<td>1948</td>
</tr>
<tr>
<td>Parry [33]</td>
<td>52/F</td>
<td>LP</td>
<td>6/60</td>
<td>24 days</td>
<td>56 days</td>
<td>Slight blurring of disc</td>
<td>Unknown</td>
<td>Optic atrophy, Central scotoma, Abn color v/a</td>
<td>1948</td>
</tr>
<tr>
<td>Sasso [34]</td>
<td>4/F</td>
<td>20/50</td>
<td>Normal</td>
<td>10 days</td>
<td>19 days</td>
<td>Disc edema and blurring, Peripapillary hemorrhage, Exudate</td>
<td>Cortisone</td>
<td>Normal fundus</td>
<td>1958</td>
</tr>
<tr>
<td>Ramseal [19]</td>
<td>78/F</td>
<td>LP</td>
<td>6/18</td>
<td>120 days</td>
<td>455 days</td>
<td>Blurred disc margin</td>
<td>Topical/subconjunctival steroids</td>
<td>Defined disc margin</td>
<td>1967</td>
</tr>
<tr>
<td>Monroe [35]</td>
<td>9/M</td>
<td>CF</td>
<td>CF</td>
<td>14 days</td>
<td>30 days</td>
<td>Disc edema with hemorrhage</td>
<td>Topical steroids</td>
<td>Disc margin sharp, Hemorrhage resolved</td>
<td>1979</td>
</tr>
<tr>
<td>Carroll [36]</td>
<td>55/M</td>
<td>CF</td>
<td>6/9</td>
<td>9 days</td>
<td>28 days</td>
<td>Central scotoma, APD, Abn VEP, EOG and color v/a</td>
<td>Unknown</td>
<td>Improved but abn VEP, EOG and color v/a</td>
<td>1979</td>
</tr>
<tr>
<td>Schmidt [4]</td>
<td>73/F</td>
<td>LP</td>
<td>1/60</td>
<td>28 days</td>
<td>240 days</td>
<td>Thickening of optic nerve, Central scotoma, Disc edema, Abn amplitude on VEP, APD</td>
<td>Systemic steroids, Vidaribine ointment</td>
<td>Optic nerve atrophy, Central scotoma</td>
<td>1983</td>
</tr>
<tr>
<td>Scharf [37]</td>
<td>73/M</td>
<td>CF</td>
<td>NLP</td>
<td>10 days</td>
<td>365 days</td>
<td>+APD, Optic nerve edema</td>
<td>Systemic steroids</td>
<td>Optic atrophy</td>
<td>1987</td>
</tr>
<tr>
<td>Tunis [17]</td>
<td>19/M</td>
<td>NLP</td>
<td>CF</td>
<td>24 days</td>
<td>90 days</td>
<td>Altitudinal scotoma, Absent VEP, APD</td>
<td>Systemic steroids</td>
<td>Optic atrophy</td>
<td>1987</td>
</tr>
<tr>
<td>Atmaca [38]</td>
<td>58/M</td>
<td>CF</td>
<td>NLP</td>
<td>9 days</td>
<td>90 days</td>
<td>APD, Disc edema, Hyperfluorescent optic disc, CRAO</td>
<td>Systemic steroid, Periocular steroid</td>
<td>Optic atrophy</td>
<td>1992</td>
</tr>
<tr>
<td>Gunduz [23]</td>
<td>48/M</td>
<td>0.1</td>
<td>0.4</td>
<td>5 days</td>
<td>90 days</td>
<td>Central scotoma, APD, Abn VEP</td>
<td>Topical steroid, Acyclovir ointment</td>
<td>Systemic steroid, B vitamins</td>
<td>1994</td>
</tr>
<tr>
<td>Menon [39]</td>
<td>48/F</td>
<td>LP</td>
<td>LP</td>
<td>30 days</td>
<td>90 days</td>
<td>Absent VEP, APD, Central scotoma</td>
<td>Systemic steroid, Known</td>
<td>Temporal disc pallor, Decreased VEP</td>
<td>1995</td>
</tr>
<tr>
<td>Deane [40]</td>
<td>73/M</td>
<td>HM</td>
<td>20/600</td>
<td>150 days</td>
<td>365 days</td>
<td>Disc edema, Splinter hemorrhages Abn VEP, Central scotoma, Abn color v/a, APD</td>
<td>Intravenous acyclovir and steroid</td>
<td>Improved visual field, Optic atrophy</td>
<td>1995</td>
</tr>
<tr>
<td>Deane [40]</td>
<td>73/M</td>
<td>20/200</td>
<td>20/80</td>
<td>42 days</td>
<td>365 days</td>
<td>Disc edema, Splinter hemorrhages, Abn VEP, Central scotoma, Abn color v/a</td>
<td>Intravenous acyclovir and steroid</td>
<td>Improved visual field, Optic atrophy</td>
<td>1995</td>
</tr>
<tr>
<td>Dhar [30]</td>
<td>30/F</td>
<td>NLP</td>
<td>5/60</td>
<td>9 days</td>
<td>28 days</td>
<td>APD, Disc edema, Peripapillary hemorrhage, Exudates, Absent VEP, Abn color v/a</td>
<td>Systemic Acyclovir, Systemic steroid</td>
<td>Improved color and contrast v, VF scotoma, Enlarged blind spot</td>
<td>1996</td>
</tr>
<tr>
<td>Mori [41]</td>
<td>50/F</td>
<td>NLP</td>
<td>20/33</td>
<td>42 days</td>
<td>240 days</td>
<td>APD, Central scotoma</td>
<td>Systemic acyclovir, Systemic steroid, SGB</td>
<td>Optic atrophy, Disc pallor, Abn color v/a</td>
<td>1997</td>
</tr>
<tr>
<td>Wang [42]</td>
<td>72/M</td>
<td>2/60</td>
<td>6/8.6</td>
<td>14 days</td>
<td>90 days</td>
<td>APD, Disc edema, VF constricted, Late staining disc, Enhanced optic nerve sheath on MRI</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Normal visual field</td>
<td>2000</td>
</tr>
<tr>
<td>Wang [42]</td>
<td>69/M</td>
<td>6/15</td>
<td>6/6.7</td>
<td>12 days</td>
<td>30 days</td>
<td>APD, Central scotoma, Enhanced optic nerve on MRI, Abn color v/a</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Resolution of APD and color deficit</td>
<td>2000</td>
</tr>
<tr>
<td>Zaal [43]</td>
<td>0.3</td>
<td>7 days</td>
<td>180 days</td>
<td>Optic neuritis</td>
<td>Systemic Acyclovir</td>
<td>Stable vision loss</td>
<td>2003</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saenz-Frances [44]</td>
<td>65/F</td>
<td>20/80</td>
<td>20/50</td>
<td>Onset</td>
<td>7 days</td>
<td>Disc edema, Peripapillary hemorrhage</td>
<td>Systemic valacyclovir, Topical acyclovir</td>
<td>Resolved disc edema, Altitudinal scotoma</td>
<td>2007</td>
</tr>
<tr>
<td>Hong [45]</td>
<td>6/F</td>
<td>0.4</td>
<td>0.8</td>
<td>7 days</td>
<td>30 days</td>
<td>Disc edema, Hyperfluorescent optic disc</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Normal fundus</td>
<td>2010</td>
</tr>
<tr>
<td>Vitor [46]</td>
<td>74/F</td>
<td>CF</td>
<td>20/60</td>
<td>45 days</td>
<td>10 days</td>
<td>Disc edema, Hyperfluorescent optic disc, Abn VF and VEP</td>
<td>Systemic acyclovir, Topical steroid</td>
<td>Disc pallor</td>
<td>2011</td>
</tr>
<tr>
<td>Gupta [47]</td>
<td>34/F</td>
<td>20/200</td>
<td>20/40</td>
<td>300 days</td>
<td>300 days</td>
<td>Optic neuritis</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Resolution of optic neuritis</td>
<td>2011</td>
</tr>
<tr>
<td>Gupta [47]</td>
<td>33/F</td>
<td>20/800</td>
<td>20/80</td>
<td>300 days</td>
<td>300 days</td>
<td>Optic neuritis</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Resolution of optic neuritis</td>
<td>2011</td>
</tr>
<tr>
<td>Our Study</td>
<td>62/M</td>
<td>LP</td>
<td>LP</td>
<td>6 days</td>
<td>30 days</td>
<td>APD, Optic disc edema, Central scotoma, Abn color v/a, APD</td>
<td>Systemic acyclovir, Systemic steroid</td>
<td>Disc pallor, Central scotoma</td>
<td>2011</td>
</tr>
</tbody>
</table>

V/A=vision; NLP=no light perception; LP=light perception; HM=hand motion; FC=finger counting; VEP=visual evoked potential; VF=visual field; SGB=stellate ganglion block; EOG=electrooculogram; APD=afferent papillary defect; M=male; F=female; ON=optic neuritis; Abn=abnormal; CRAO=central retinal artery occlusion
Table 2. Characteristics extracted from table 1. Note the significant decrease in both legal blindness and optic atrophy in the acyclovir treated group compared to the non-acyclovir treated group. Legal blindness based on logMAR 1.0 or 20/200.

<table>
<thead>
<tr>
<th></th>
<th>Acyclovir treated group (n=13)</th>
<th>Non-acyclovir treated group (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Age (years)</td>
<td>53</td>
<td>58</td>
</tr>
<tr>
<td>Gender</td>
<td>6 Male/7 Female</td>
<td>6 Male/7 Female</td>
</tr>
<tr>
<td>Rate of legal blindness</td>
<td>23%</td>
<td>69%</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>31%</td>
<td>69%</td>
</tr>
</tbody>
</table>

Figure 3. LogMar visual acuity equivalent data points for each case report selected. Most of the cases treated with acyclovir were also treated with systemic steroids. Of those not treated with systemic acyclovir, about half received systemic steroids. Most of the cases treated with acyclovir maintained stable useful vision as shown in the trendline analysis.

logMAR visual acuity of 1.77 (n=6). Most cases treated with systemic acyclovir were also given systemic steroids (n=10). This subset had a logMAR visual acuity of 0.72 (figure 4). Legal blindness (logMAR 1.0) occurred in 23% of the acyclovir treatment group compared to 69% of the non-acyclovir treatment group (table 2).

Discussion

Herpes zoster related optic neuritis is a rare complication occurring more often in the elderly and immunosuppressed as cell-mediated immunity falls. Because of its uncommon nature, the incidence is unknown but estimated at far less than 1%. Advances in technology however may indicate that inflammation of the optic nerve occurs more often than its clinical presentation. Wenkel et al. performed histological examination and immunohistochemistry of 9 ocular specimens with a history of HZO. Their evaluation found over 50% of these eyes had evidence of optic neuritis confirmed by viral DNA detected at the optic disc. In this study viral DNA was identified in many other structures.
including: the anterior chamber, corneal stroma, episclera, posterior ciliary nerves and arteries, and meninges causing a granulomatous inflammation. Viral DNA was even identified in some specimens up to 10 years after the onset of HZO. This may account for the chronic recurrent nature of the disease. Naumann et al. also discussed the histopathology of 21 enucleated eyes after HZO. Seven of these eyes demonstrated evidence of optic neuritis although none had clinical symptoms before enucleation. In this study inflammation was also found around the posterior ciliary nerves and vessels with patchy necrosis of the iris suggestive of generalized ischemia.

HZO optic neuritis may be unilateral, bilateral, intraocular or retrobulbar. The optic nerve may become involved by several different mechanisms. Direct extension, hematogenous, cerebral spinal fluid and transneuronal spread have all been implicated. Studies by Wenkel et al. and Naumann et al. confirmed vascular inflammation resulting in ocular ischemia. Cerebral spinal fluid may allow spread of the herpetic family. Transneuronal spread has been implicated as evident on MRI. Concurrent MRI T2 signal enhancement of the optic nerve, tracts, lateral geniculate body, optic radiations and calcarine fissure support this theory. Direct extension of the virus through the cavernous sinus also occurs as evident in herpes zoster associated orbital apex syndrome. Despite the lack of histological evidence in our case, papillitis secondary to direct viral induced neuritis and vasculitis is suspected.

Acute optic neuritis usually develops as a post-herpetic complication. In this analysis the onset of optic nerve disease occurred from 5 days to 5 months but the average time was about 4 weeks (figure 5). This confirms previous reported post-herpetic onset times ranging from 2 to 4 weeks. Time to resolution was similar in each group. This data was probably confounded by variability in patient follow up, meaning the exact time of resolution was probably overestimated. The severity and course of ocular inflammatory complications depends on the interaction between reactivated virus, the immune response elicited and the timing and efficacy of anti-viral therapy.

Before the development of anti-viral therapy, herpes zoster related optic neuritis had a dismal outcome. Most ended with less than 20/200 vision secondary to optic atrophy. This study confirmed that without acyclovir, visual prognosis is poor and optic atrophy likely (figures 3 and 4). Table 2 shows that legal blindness is 3 times higher when anti-viral therapy is not used to treat optic neuritis. Acyclovir has become standard of care for the treatment of HZO.

Acyclovir is a specific antiviral agent against herpetic infections that inhibits viral DNA polymerase. High doses of acyclovir are needed to treat the virus because acyclovir has a poor bioavailability and the zoster virus is less susceptible than other herpetic infections to this medication. A dose of 800 mg of oral acyclovir 5 times daily or 10 mg per kilogram intravenously every 8 hours has been shown by multiple studies to be effective. The duration of treatment has also been studied. These studies have proven a 7 day course to be effective and that longer durations have no additional benefit. The benefits of acyclovir are best achieved if treatment is begun within 72 hours of skin examination. At the appropriate dose, acyclovir not only decreases viral shedding but also decreases the severity of ocular sequelae.

Our study supports evidence of acyclovir’s efficacy (figures 3 and 4). Visual outcomes are significantly improved when systemic acyclovir is used to treat herpes zoster related optic neuritis. A gain of more than 3 lines on a Snellen equivalent eye chart was achieved using this treatment when compared to non-treated groups. The prevalence of optic atrophy and legal blindness both fell by over 50% with the advent and implication of acyclovir treatment (table 2). Steroids have also commonly been used in adjuvant to acyclovir (table 1). Steroids alone do not have promising visual outcomes but may help decrease the vasculitis associated with HZO related optic neuritis when used with acyclovir (figure 4). Our patient despite treatment with acyclovir and steroid did not regain useful vision. Perhaps because acyclovir therapy was delayed and significant viral load had already been established, damage was inevitable. Despite our best efforts however, herpes zoster related optic neuritis frequently leaves the patient with some amount of permanent vision loss.
Figure 4. A comparison of visual acuities over selected treatment groups. Most of the cases treated with acyclovir were also treated with systemic steroids. The visual acuity benefits are attributed mostly to acyclovir as the steroid only group performed poorly.

Figure 5. Estimated Optic Neuritis Onset and Recovery. Mean onset and recovery were comparable. Recovery time is based on patient follow up and probably is overestimated.

Prevention would be ideal in fending this virus. To date, we cannot prevent herpes zoster reactivation. There is no rapid zoster test to catch it during its prodromal stage. Vaccines however may provide a boost in cell-mediated immunity for the elderly and immunocompromised so the body can maintain at bay this latent virus.\textsuperscript{2,3}

**Conclusion**

Systemic acyclovir has dramatically reduced the morbidity of ocular complications caused by HZO. Any ocular structure can be involved. The optic nerve is less commonly affected by HZO resulting in papillitis, neuroretinitis or retro-bulbar neuritis. When herpes zoster does infect the optic nerve the consequences can be devastating. High dose systemic acyclovir is currently the treatment of choice for this condition and can improve chances of good visual outcomes as well as quality of life in immunocompetent persons.

**Comments**

No potential conflict of interest relevant to this article was reported. This study met IRB exemption requirements because it involves the collection of existing data, documents and records. The information is expressed in such a manner that subjects cannot be identified directly or through identifiers.
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